Dissertation On

A Study of The Relationship of Poststroke Depression to Location of Inter Hemispheric and Intra Hemispheric Lesion

submitted in partial fulfilment of
requirements for

M.D. DEGREE BRANCH XVI GERIATRIC MEDICINE

of

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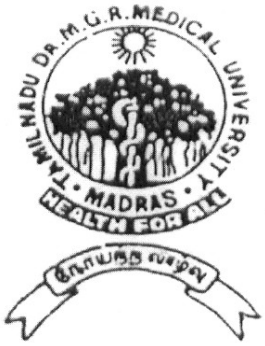

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF THE RELATIONSHIP OF POSTSTROKE DEPRESSION TO LOCATION OF INTER HEMISPHERIC AND INTRA HEMISPHERIC LESION"** submitted by **Dr.K.ANUPAMA,** appearing for Part II M.D. Branch XVI Geriatric Medicine Degree Examination in March 2008 is a bona fide record of work done by her under my direct audience and supervision in partial fulfillment of regulations of the Tamil Nadu Dr.M.G.R.Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF THE RELATIONSHIP OF POSTSTROKE DEPRESSION TO LOCATION OF INTER HEMISPHERIC AND INTRA HEMISPHERIC LESION"** is done by me at Madras Medical College & Govt. General Hospital, Chennai during 2005-2008 under the guidance and supervision of **Prof. Dr.B.K.KRISHNASWAMY, M.D.**

The dissertation is submitted to The Tamilnadu **Dr.M.G.R.Medical University** towards the partial fulfillment of requirements for the award of M.D. Degree (Branch XVI) in Geriatric Medicine.

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CONTENTS

INTRODUCTION

Cerebrovascular disease is one of the most common life threatening problems among elderly and it ranks only behind heart disease and cancer as the third leading cause of death. Among survivors 75% are left with physical or intellectual impairments of sufficient severity to limit their vocational capacity. Depression is the most common emotional disorder associated with cerebrovascular disease. In a basic science framework, it has been reported that stroke might cause depression by means of lesion related disruption of catecholamine pathways. Initial studies found an increased risk of PSD with left brain lesions. The available data suggest that PSD is not a transient reaction to the consequences of stroke but rather a long standing disorder with a natural course of less than one year for major depression and a more variable course for minor depression. Some major depressions however last more than three years and some minor depressions evolve into major depression and may last for several years. There is a significant influence of PSD on language functioning. It also adversely affects the extent of recovery made with respect to activities of daily living. PSD has been shown to increase the mortality rate following stroke.

This study aims to find the correlation between poststroke depression and inter and intra hemispheric lesion location.

AIM OF THE STUDY

1. To find out if there is any correlation between poststroke depression and the side of lesion.

2. To find out if there is a correlation between poststroke depression and the site of lesion in both right and left cerebral hemispheres.

3. To study the relationship between select risk factors for stroke and poststroke depression.

4. To find out if there is a correlation between the severity of poststroke depression and the duration from onset of stroke.

REVIEW OF LITERATURE

1. **Lesion location and poststroke depression.** Philip L. P. Morris et al, Journal of Psychiatry, Vol.8, No.4, Fall 1996, 399-403.

Purpose

To examine whether stroke lesions involving left hemisphere prefrontal or basal ganglia structures are associated with PSD.

Methods

Forty one consecutive consenting patients with a first – ever, stroke and a single, small (less than 20% of total brain volume) infarct or hemorrhage on CT Scan were examined approximately eight weeks after the stroke.

Results

Patients with left hemisphere prefrontal or basal ganglia lesions did not differ from patients with other left hemisphere or right hemisphere lesions with respect to age, sex, marital status, functional performance, physical disability, presence of a past personal or family history of mood disorder, lesion type or lesion volume. 37% had a diagnosis of clinical depression.

Discussion

The main finding of this study was that left hemisphere lesions involving the prefrontal cortex or basal ganglia structures were associated with PSD.

2. **Mood disorders after stroke and their relation to lesion location study.** Allan House et al. Brain Vol. 113 No.4 Pg 1113-1129, 1920.

Purpose

73 consecutive patients with a first- ever stroke, who had a CT scan which showed a neurologically appropriate single stroke lesion and who did not have a psychiatric disorder in the year preceding the stroke, were followed up.

Results

No evidence that left sided lesions were associated with more severe or persistent depressive symptoms. There was a weak correlation between mood symptom scores and the proximity of the stroke lesion to the frontal pole of the hemisphere.

Conclusion

No difference occurred between right and left hemisphere strokes and in the relationship between lesion distribution and mood symptoms.

3. **Two year longitudinal study of poststroke mood disorders; dynamic changes in correlates of depression at one and two years.** RM Parikh et al. Stroke 1987; 18: 579-584.

Purpose

To assess factors associated with poststroke depression and examine changes in the strength of these relations over the two years of follow-up.

Methods

103 inpatients at University of Maryland Hospital were examined. 86 patients were interviewed again at least once during the two year follow up study.

Results

During acute hospitalization, the most severe impairment in functional physical activity or cognition was associated with the most severe depression. Patients with poorer social support were more depressed than these with better social supports.

Conclusion

Left hemisphere lesion location had the strongest correlation with severity of depression for the first year after stroke. Functional physical impairment had a significant correlation with depression throughout the entire study period. Cognitive impairment was significantly correlated with depression for the first six months after stroke.

4. **Functional Impairment Associated With Acute PSD** - Rajamannar Ramasubhu et al. J. Neuro. Psychiatry Clin. Neuro Sci. 10 : 26-33 February 1998.

Purpose

To examine the independent association of depression following acute stroke with impairment in activities of daily living.

Methods

Patients with a first-ever stroke were examined. ADL assessment was performed by using the Barthel Index.

Results

Patients with depression had significantly more impaired functioning than nondepressed patients.

Conclusion

Depression is not simply a reaction to the severity of functional impairment. Of all the factors, depression may be the only treatable condition that independently affects the functioning of patients following acute stroke. Early recognition and effective treatment of depression following stroke might optimize rehabilitation potential.

5. **Qualitative & quantitative measurement of depression in veterans recovering from stroke** - Christine L. Williams et al., Journal of Rehabilitation research and development Vol.42, No.3, May – June 2005, Pg. 277-290.

Purpose

To analyse qualitative and quantitative data from inter views, GDS scores and patient records to evaluate PSD.

Methods

112 respondents from 5 Department of veterans affairs who were hospitalized for a new stroke were chosen.

Methodological triangulation was used to overcome bias that occurs when data are obtained for a single method.

Results

At one month following discharge from the hospital, participants had a mean score on the GDS of 8.76. GDS was found to be reliable. Overall prevalence of depression was 35%.

Conclusion

Undertreatment of depression occurs in post stroke population. GDS excludes the vegetative signs of depression and may not adequately capture symptoms of depression in those who focus on somatic complaints

6. **Comparison of six depression rating scales in geriatric stroke patients -** Bagrell et al. Stroke vol 20, 1190-1194.

Three self-rating scales (GDS, Zung scale and the Center for Epidemiologic Studies Depression Scale), were compared with three examiner – rating scales (Hamilton Rating Scale, the Comprehensive

Psychopathological Rating Scale Depression, and the Cornell scale) to see which was best for elderly stroke patients. GDS, Zung scale and the Comprehensive Psychopathological Rating scale Depression had the highest sensitivity and the Zung scale had the highest positive predictive value. With regard to internal consistency, sensitivity and predictive value the best self rating scales were GDS and Zung scales and the best examiner rating scale was the Comprehensive Psychopathological Rating Scale Depression.

7. **A Reappraisal of Poststroke Depression, Intra and Inter - Hemispheric Lesion location using meta analysis.** K. Narushima et al. J. Neuropsychiatry Clin. Neurosci. 15 : 422-430, Nov. 2003.

All studies that examined the correlation between PSD and lesion location were included. Journal articles between January, 1981 and December 2000 were screened.

Results showed that there was a significant inverse correlation between the severity of depression and distance of the lesion from the frontal pole among patients with left hemisphere stroke but not among patients with right hemisphere stroke. This study supports the hypothesis that risk of PSD is related to the location of brain injury.

8. **Are depressive symptoms nonspecific in patients with acute stroke?** J.P. Fedoroff, SE Starkstein, RM Parikh, TR Price & RG Robinson. Am. J. Psychiatry 1991; 148 : 1172-1176.

Purpose

Major depression might be overdiagnosed in stroke patients because of changes in appetite,

sleep or sexual interest while others suggest that it may be underdiagnosed in stroke patients who deny symptoms of depression because of anosognosia, neglect or prosody. The authors goal was to determine how frequently depressive symptoms occur in acute stroke patients with and without depressed mood to estimate how often diagnostic errors of inclusion or exclusion can be made.

Methods

Rate of autonomic and psychological symptoms of depression in 205 patients who were hospitalized for acute stroke were examined. 41% had depressed mood, 59% had no mood disturbance. 54% of the 85 patients with depressed mood were assigned the DSM IV diagnosis of major depression.

Results

The 120 patients without mood disturbance had a mean of one autonomic symptom but the 85 patients with depressed mood had a mean of almost four. Lightening the diagnostic criteria to account for one more non specific autonomic symptom decreased the number of patients with major depression by only three; adding two more criteria decreased the number by only five.

Conclusion

Both autonomic and psychological depressive symptoms are strongly associated with depressed mood in acute stroke patients.

9. **The impact of post strokedepression on recovery in ADL over a two year follow up.** R.M. Parikh, R.G. Robinson, J.R. Lipsey et al. Archives of Neurology Vol. 47, No. 7, July 1990.

The impact of clinically diagnosed depression on recovery in activities of daily living over a 2 year follow up was examined in a prospective study of 63 stroke patients. Although impairment in ADL, neurologic diagnoses and findings, lesion location and volume as measured on computed tomographic scan, demographic variables, cognitive impairment and social functioning were comparable between depressed and nondepressed patients during their acute hospitalization, the two groups had different patterns of recovery in activities of daily being. At 2 years after suffering a stroke, patients with in-hospital diagnosis of depression were significantly more impaired in both physical activities and language functioning than were nondepressed patients. This study emphasises the need for early recognition and treatment of post stroke depression.

STROKE

A stroke or cerebrovascular accident is defined by the abrupt onset of a neurological deficit that is attributable to a focal vascular cause. The WHO defines stroke as “rapidly developing clinical signs of focal (at times global) disturbance of cerebral function, lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin”.

This definition includes signs and symptoms suggestive of

- Ischemic stroke
- Hemorrhagic stroke (intracerebral or subarachnoid)

INCIDENCE IN INDIA

A WHO study quotes the incidence of stroke in India to be around 130 per 100,000 population every year and says about 20% of heart patients are susceptible to it. It is the most common cause of disability and dependence with more than 70% of stroke survivors remaining vocationally impaired and more than 30% requiring assistance for activities for daily living.

RISK FACTORS FOR STROKE

- Age
- Hypertension
- Atrial fibrillation
- Diabetes Mellitus
- Smoking
- Hyperlipidemia
- Carotid artery stenosis

Common Causes of Ischemic Stroke

Thrombosis
- Lacunar stroke
- Large vessel thrombosis
- Dehydration

Embolic occlusion

Artery to artery
- Carotid bifurcation
- Aortic arch
- Arterial dissection

Cardio embolic
- Atrial fibrillation
- Myocardial infarction
- Dilated cardiomyopathy
- Valvular lesions
  - Mitral stenosis
  - Mechanical valve
  - Bacterial endocarditis
- Paradoxical embolus
  - Atrial septal defect
  - Patent foramen ovale
- Atrial septal aneurysm
- Spontaneous echo contrast

Causes of Intracerebral hemorrhage

- Hypertension
- Aneurysm
- AV malformation
- Amyloid angiopathy
- Head trauma
- Transformation of prior ischemic infarction
- Metastatic brain tumour
- Coagulopathy
- Drug
- Cavernous angiomas
- Ductal arteriovenous fistula
- Capillary telangiectasis

Stroke Syndromes

1. Large vessel stroke within the anterior circulation.
2. Large vessel stroke within the posterior circulation.
3. Small vessel disease of either vascular bed.

STROKE WITHIN THE ANTERIOR CIRCULATION

Middle cerebral artery

Signs and symptoms – structures involved.

- **Paralysis of contralateral face, arm and leg; sensory impairment over the same area**. Somatic motor area for face and arm and the fibres descending from the leg area to enter the corona radiata and corresponding somatic sensory system.
- **Motor aphasia -** Motor speech area of dominant hemisphere.
- **Central aphasia, word deafness, anomia, jargon speech, sensory agraphia, acalculia, alexia, finger agnosia, right-left confusion** - Central, suprasylvian speech area and parieto occipital cortex of the dominant hemisphere.
- **Conduction aphasia** - Central speech area.
- **Apractognosia of nondominant hemisphere, anosognosia, hemiasomatognosia, dressing apraxia, constructional apraxia** – Nondominant parietal lobe
- **Homonymous hemianopia** - Optic radiation.
- **Paralysis of conjugate gaze to opposite side** - Frontal contraversive field.

Anterior cerebral artery

Signs & Symptoms : structures involved

- **Paralysis of opposite foot and leg** - Motor leg area.
- **Cortical sensory loss of foot and leg** - Sensory area for foot and leg.
- **Urinary incontinence** - Sensorimotor area in paracentral lobule.
- **Contralateral grasp reflex, sucking reflex, gegenhalten**. - medial surface of posterior frontal lobe.
- **Abulia, slowness, delay -** uncertain
- **Gait apraxia**. - frontal cortex near leg motor area
- **Dyspraxia of left limbs, tactile aphasia in left limbs -** Corpus callosum

Anterior choroidal artery – contralateral hemiplegia, hemianesthesia , homonymous hemianopia.

Internal carotid artery and common carotid artery – varying picture of middle cerebral artery and anterior cerebral artery territory involvement and amaurosis fugax.

STROKE WITHIN THE POSTERIOR CIRCULATION

Posterior Cerebral Artery

Signs & Symptoms : Structures involved

Peripheral territory

- **Homonymous hemianopia. -** Calcarine cortex
- **Bilateral homonymous hemianopia, cortical blindness, awareness or denial of blindness, apraxia of ocular movements -** Bilateral occipital lobe with parietal lobe
- **Verbal dyslexia without agraphia, color anomia** - dominant calcarine lesion and posterior part of corpus callosum
- **Memory defect -** Hippocampus
- **Prosapagnosia -** Nondominant calcarine and lingual gyrus
- **Simultagnosia , hemivisual neglect -** Dominant visual cortex and contralateral hemisphere.

Central territory

- **Thalamic syndrome -** Posteroventral nucleus of thalamus
- **Claude's syndrome -** Dentatothalamic tract
- **Weber's syndrome -** Third nerve and cerebral peduncle
- **Contralateral hemiplegia -** cerebral peduncle
- **Paresis of vertical eye movement, skew deviation, sluggish pupillary responses to light,**

miosis, ptosis - Supranuclear fibres to third nerve, interstitial nucleus of Cajal, nucleus of Darkschewitsch.

Vertebral / posterior inferior cerebellar arteries

Medial medullary syndrome

On side of lesion

- **Paralysis with atrophy of half the tongue** - ipsilateral twelfth nerve.

On side opposite lesion

- **Paralysis of arm and leg sparing face, impaired tactile and proprioception over half the body** - contralateral pyramidal tract and medial lemniscus.

Lateral medullary syndrome

On side of lesion

- **Pain, numbness, impaired sensation over half the face** – descending tract and fifth nerve nucleus.
- **Ataxia of limbs, falling to side of lesion** - uncertain
- **Nystagmus, diplopia, oscillopsia** - Vestibular nucleus
- **Horner's syndrome** – descending sympathetic tract

- **Dysphagia, hoarseness, paralysis of palate and vocal cord, diminished gag reflex** - ninth and tenth nerves
- **Loss of taste** – nucleus and tractus solitarius
- **Numbness of ipsilateral arm, trunk or leg** – cuneate and gracile nuclei

On side opposite lesion

- **Impaired pain and thermal sense over half the body** - Spinothalamic tract.

Basilar Artery syndrome

Combination of various brainstem syndromes plus those arising in the posterior cerebral artery distribution.

Imaging Studies

Computed Tomographic Scans

To identify or exclude hemorrhage as a cause of stroke. The infarct may not be seen reliably for 24 to 48 h. It may fail to show small ischemic infarcts in posterior fossa; small infarcts on the cortical surface may also be missed.

Magnetic Resonance Imaging

Less sensitive for acute blood products than CT. Diffusion- weighted imaging is more sensitive for early brain infarction as is FLAIR (fluid attenuated inversion recovery) imaging.

Cerebral Angiography

Gold standard for quantifying atherosclerotic stenosis of cerebral arteries and for identifying other pathologies - aneurysm, vasospasm, intraluminal thrombi.

MOOD DISORDERS

Mood disorders are characterized by a disturbance in the regulation of mood.

They are subdivided into

1. Depressive disorders
2. Bipolar disorders
3. Depression in association with medical illness or alcohol and substance abuse.

Depressive disorders

Major Depression

Major depression is defined as depressed mood on a daily basis for a minimum duration of two weeks. An episode may be characterized by sadness, indifference, apathy or irritability and is usually associated with changes in sleep patterns, appetite, and weight, motor agitation or retardation, fatigue, impaired concentration and decision-making , feelings of shame or guilt and thoughts of death or dying. Patients with depression have a profound loss of pleasure in all enjoyable activities, exhibit early morning awakening, feel that the dysphoric mood state is qualitatively different from sadness, and often notice a diurnal variation in mood (worse in morning hours).

Approximately 15% of the population experiences a major depressive episode at some point in life, and 6-8% of all outpatients in primary care settings satisfy diagnostic criteria for the disorder**.**

Risk Factors

- History of prior episodes of depression
- Family history of depressive disorder especially in first degree relative
- History of suicide attempts
- Female gender
- Age of onset before age 40
- Postpartum period
- Co-morbid medical illness
- Absence of social support
- Negative stressful life events
- Active alcohol or substance abuse
- Profound hopelessness

DSM-IV-TR Criteria for Major Depressive Episode

A. Five (or more)of the following symptoms have been present during the same 2 week period and represent a change from previous functioning : at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure. Note: Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report or observation made by others.

2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day.

3. Significant weight loss when not dieting or weight gain or decrease or increase in appetite nearly every day.

4. Insomnia or hypersomnia nearly every day.

5. Psychomotor agitation or retardation nearly every day.

6. Fatigue or loss of energy nearly every day.

7. Feelings of worthlessness or inappropriate guilt nearly every day.

8. Diminished ability to think or concentrate , or indecisiveness, nearly every day.

9. Recurrent thoughts of death, recurrent suicidal ideation without a specific plan.

B. The symptoms do not meet criteria for a mixed episode.

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The symptoms are not due to the direct physiologic effect of a substance or a general medical condition.

E. The symptoms are not better accounted for by bereavement, the symptoms persist for >2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

Dysthymic disorder consists of a pattern of chronic (at least 2 years), ongoing, mild depressive symptoms that are less severe and less disabling than those found in major depression; the two conditions are sometimes difficult to separate, however, and can occur together("double depression").

Minor depression is used for individuals who experience at least 2 depressive symptoms for 2 weeks but do not meet the full criteria for major depression.

DSM-IV_TR Criteria for Severity, Psychotic, and Remission Specifiers for current Major Depressive Episode

Mild : few, if any, symptoms in excess of those required to make the diagnosis. Symptoms result in only minor impairment in occupational functioning or in usual social activities or relationships with others.

Moderate : Symptoms or functional impairment between mild and severe.

Severe without psychotic features : Several symptoms in excess of those required to make the diagnosis. Symptoms markedly interfere with occupational functioning or with usual social activities or relationships with others

Severe with psychotic features : Delusions or hallucinations. If possible, specify whether the psychotic features are mood-congruent or mood-incongruent.

Mood-congruent psychotic features : Delusions or hallucinations whose content is entirely consistent with the typical depressive themes of personal inadequacy, guilt, disease, death, nihilism, or deserved punishment.

Mood-incongruent psychotic features : Delusions or hallucinations whose content does not involve typical depressive themes of personal inadequacy, guilt, disease, death, nihilism, or deserved punishment. Included are such symptoms as persecutory delusions (not directly related to depressive themes), thought insertion, thought broadcasting, and delusions of control.

In partial remission : Symptoms of a major depressive episode are present, but full criteria are not met, or there is a period without any significant symptoms of a major depressive episode lasting less than 2 months after the end of the major depressive episode.

In full remission : During the past 2 months, no significant signs or symptoms of the disturbance were present.

Unspecified.

Major Depressive Disorders (MDD) in the Elderly

Major depressive disorders have important medical, social and financial consequences. They cause suffering to patients and their families, exacerbate medical illnesses, cause disability, and require expensive support systems. They can be effectively treated in most cases. Therefore it is crucial to identify them and to treat them appropriately.

Geriatric depression is unrecognized. Clinicians and patients often attribute depressive symptoms to the aging process. Another reason is that older persons emphasize somatic symptoms and underreport depressed mood. Geriatric depression often occurs in the context of medical or neurological brain diseases whose symptoms are similar to the symptoms of depression. In some cases, the overlap of symptomatology is such that depression can only be diagnosed after successful antidepressant treatment. Symptoms of depression may simulate dementia with concentration difficulties, memory loss and distractibility. Commonly MDD and dementia co occur.

The overall prevalence of major depression among persons 65 years of age or older is estimated to be 1.4% in women and 0.4% in men, with a overall prevalence of 1 %. This prevalence rate is approximately one-fourth that of younger adults.

Comorbidity Patterns -General Medical Conditions

Whereas a 4 to 5% current prevalence rate of MDD exists in community samples, symptoms of depression are found in 12-36% of patients with a general medical condition. The possible relationships that could exist between depression and a general medical condition are as follows-

1. Depression is biologically caused by the general medical condition.
2. Individual who carries a genetic vulnerability to MDD manifests the onset of depression triggered by the general medical condition.
3. Depression is psychologically caused by the general medical condition.
4. No causal relationship exists between the general medical condition and mood disorder.

Suicide Risk

Suicide is almost twice as frequent in older adults as in the general population. Aging reduces suicide attempts but increases their lethality. The most common precipitants of suicide in older individuals are physical illness and loss, whereas problems with employment, finances, and family relationships are more frequent precipitants in younger adults. Severity of depression is the strongest predictor of the course of suicidal ideation.

POSTSTROKE DEPRESSION

Prevalence ranges from 25-50% (1-3) in various studies. For the diagnosis of major depression due to stroke, the DSM – IV TR criteria are used, excluding the criteria which precludes an organic factor .(4)

CRITERIA

Five or more of the following symptoms have been present during the same 2 week period and represent a change from previous functioning; at least one of the symptoms is either 1 or 2.

1. Depressed mood most of the day, nearly every day as indicated by either subjective report or observation.
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day.
3. Significant weight loss or weight gain or appetite change.
4. Insomnia or hypersomnia.
5. Psychomotor agitation or retardation, observable by others.
6. Fatigue or loss of energy.
7. Feelings of worthlessness or inappropriate guilt.
8. Diminished ability to concentrate or make decisions.
9. Recurring thoughts of death or suicidal thoughts or plans.

Varying definitions, populations, exclusion criteria, time after stroke have been postulated to account for inconsistent research findings[5,6,7,8].

No clear consensus exists on how best to define major depression after stroke or its variants. Some investigators have categorized subjects based on psychiatric interview and DSM based definitions, others have used psychiatric rating scales. Different emphasis is placed on various domains of depressive illness across scales with interpretation of somatic symptoms constituting an area of controversy in poststroke depression (PSD). Scales include Hamilton rating scale for depression, Beck Depression Inventory, the Center for Epidemiologic Studies Depression Scale, the Montgomery Asberg Depression Rating Scale, the Zung Self -Rating Depression Scale and the Geriatric Depression Scale. The relative diagnostic accuracy of these tools, applied to patients with stroke is unknown.

Second, factors in the selection of the study people are critically important. Inpatient settings show higher rates of PSD than those found in community based samples. Patients with significant cognitive impairment or aphasias are excluded because of difficulty assessing mood state, but this may reduce the ability to generalize results to the broader stroke population. In many studies, only patients with first ever stroke are included; others have limited studies only to those whose brain lesions were confirmed by imaging and correlating with clinical presentation. When preexisting depression was excluded, there were lower rates of incident PSD.

Third timing of assessment of PSD has varied in different studies. In the immediate poststroke period, adjustment reactions may confound assessment. The stages that follow stroke include crises, active rehabilitation and long term adjustment phase.[6] Thus, evaluation carried out at a few weeks after stroke may identify depressive symptoms of a different nature than those found one year or more after stroke.

Also changes in regulation of affective behaviour have been noted as a frequent consequence of stroke. Instruments for primary depression are postulated to capture changes that arise from stroke, rather than from depression. Patients with aprosodic expression lack the usual language and nonverbal expressions of affective communication. A flat affect may reflect stroke injury by impairing mechanisms of expression or may be accompanied by denial of impairments and defective experience of mood. At the other extreme, post stroke pathologic crying may be misinterpreted as an expression of depressed mood.[9]

Mechanism of depression following stroke

Controversy exists about whether PSD[19] is a separate type of depressive syndrome that is specific to the post stroke state[17,18]. Some argue that if PSD were simply a reaction to the consequences of stroke, then the risk of depression should be clearly associated with the extent of functional impairment, a finding not consistently demonstrated in published reports. Stroke might cause depression by means of lesion related disruption of catecholamine pathways [10, 11, 12]. The noradrenergic and serotonergic cell bodies are located in the brainstem and send ascending projections through the medial forebrain bundle to the frontal cortex. The ascending axons then arc posteriorly and run longitudinally through the deep layers of the cortex, arborizing and sending terminal projections into the superficial cortical layers . Lesions that disrupt these pathways in the frontal cortex or the basal ganglia may affect many downstream fibers. Based on these neuroanatomical facts and the clinical findings that the severity of depression correlates with the proximity of the lesion to the frontal pole, Robinson et al., suggested that PSD may be the consequence of severe depletion of norepinephrine and/or serotonin produced by frontal or basal ganglia lesions. A greater depletion of biogenic amines in patients with right hemisphere lesions as compared to those with left hemisphere lesions could lead to a compensatory up-regulation of receptors that might protect against depression. Patients with left

hemisphere lesions may have moderate depletion of biogenic amines but without a compensatory up-regulation of serotonin receptors and therefore a dysfunction of biogenic amine systems in the left hemisphere. This dysfunction ultimately may lead to the clinical manifestations of depression[14].

Another possibility is that both the frontal dorsolateral cortex and the dorsal caudate play an important role in mediating motor, intellectual and instinctive behaviour through their connection with the supplementary motor area, temporo parietal association cortex and limbic system. A lesion of these anterior brain areas may result in low activation of motor, sensory or limbic areas and produce the autonomic and affective symptoms of depression.

Gainotti et al.[15] developed a rating scale specifically designed for affective problems in stroke. Its' domains include depressed mood, guilt, thoughts of death, suicide, vegetative disorders, apathy, anxiety, catastrophic reactions, hyperemotionalism, and diurnal variation in mood. Findings from this suggest that depression in stroke may be of a different nature than that which is sometimes called primary depression.

Risk for PSD

Lesion related factors and stroke severity

Much of the initial work on laterality of stroke found an increased risk of PSD with left brain lesions[16,27]. But not all investigators have replicated his finding. Many have found no association. Others have found right brain lesions to be highly associated with PSD.

Robinson et al.[11] and Starkstein et al.[20,21] found that major or minor PSD showed higher frequency of lesions in anterior areas of left hemisphere-left frontal dorsolateral cortex.He also found a relationship with specific subcortical lesions – basal ganglia and PSD. In right hemisphere stroke, those with frontal or parietal damage showed higher frequency of depression[22,23]. Robinson et al. [24] found an inverse correlation between the severity of depression and distance of the anterior border of the lesion from the frontal pole. Some found correlation between depression and proximity of lesion to the frontal pole in combined right and left hemisphere lesion groups [1,26], whilst others found it only with left- sided lesions [2,28].The proximity of the lesion to the frontal pole is associated strongly with severity of depression during the first 6 months poststroke [25].

Stroke severity and the extent of resulting disability is also inconsistent with the occurrence of PSD. Investigators have found depression to be more likely among persons with large lesions, severe stroke injury scores and more extensive functional disability . Others have not confirmed this.

Demographic and Personal factors

Women were twice as likely as men to have major depression. Men with major depression had greater functional impairment, whereas women with more severe depression were more likely to have cognitive impairment or prior psychiatric illness, suggesting that gender difference may modify PSD [29,30].

In the Perth Community Stroke Study, age and gender were not found to be associated with PSD. Significant associations with PSD at four months were functional impairment, nursing home residence, being divorced, and for men, a high in take of alcohol [31] before the stroke. In another study, previous stroke, previous depression, female gender, living alone and social difficulties in the six months before stroke were correlated with PSD. Prior personal history of depression also has been found by others to be associated with PSD as has a family history of depression or anxiety.

Immediate depression was best predicted by left anterior lesion location, aphasia and living alone. By three months, dependency in ADL was found to be most important. At one year and onward, having few social contacts outside immediate family members was important and at three years cerebral atrophy became predictive of PSD as well. Initial PSD may be related to neurological and organic factors, but socio demographic elements are more important in determining who goes on to develop chronic depression[9].

Tools for screening and evaluation

Reports suggest that the Geriatric Depression Scale (GDS) is satisfactory for this population[32,33]. The 15-item short form has been studied widely and validated in a number of patient groups and requires little time to administer. Elderly patients can have fewer mood and more somatic complaints which are often difficult to differentiate from stroke-related impairment. GDS considers this and so is especially situated for this population. Andersen advocates use of HDRS citing reports that find no significant elevation in the rate of diagnosis of major depression despite concerns about its somatic items. The CES-D was found to have sensitivity of 86% and specificity of 90% for PSD with a

16 point out off [34]. The PSD rating scale by Gainotti has demonstrated high interrater reliabilty[15]

Neuroendocrine markers of depression

Use of dexamethasone suppression test, which in depressed but not brain – injured persons results in lack of the usual suppression of morning cortisol after a night time dose of dexamethasone[16]. Wide variability in test characteristics for the DST have been found.

Clinical presentation of PSD

Except for minor differences the clinical presentation of PSD and idiopathic depressive illness are similar. Physical and mental slowness are more frequent in PSD; loss of interest, concentration more common in idiopathic depression. Some researchers suggest that patients with PSD exhibit more anxiety, worry and tearfulness but less guilt, worthlessness and suicidal thoughts.

Cognitive impairment is similar to the syndrome of pseudodementia seen in patients with idiopathic depression. Bolla Wilson et al. found that patients with depression and left hemisphere lesions had significant greater cognitive impairment than nondepressed patients with similar lesions matched for size and location. In patients with right hemisphere lesions, cognitive function did not differ between depressed and nondepressed groups.

Assessment of PSD

A variety of factors may hinder the diagnosis of PSD. Clinicians and family usually judge the patient`s mood on his / her outward affective expression, emotional lability and bradykinesia, but these signs are unreliable in stroke patients.

The effects of stroke may mimic depression or impair emotional communication. Patients with aphasia may be unable to report their feelings. Injury to the right hemisphere or frontal lobes may cause indifference, foolish and superficial affect and a lack of awareness, and impaired emotional communication. The dysprodic patient may appear flat, emotionally blunted and depressed; the clinician must inquire about the patient's underlying mood state. Patients with heightened emotionalism may / may not have a full depression syndrome. Patients with bilateral frontal lobe or sub cortical injury may have marked emotional lability or pseudobulbar palsy.

The clinician must observe for signs of depression, apathy, irritability and inquire about the patients' appetite, weight loss, sleep patterns and level of sexual interest and activity.

Treatment of PSD

Treatment among frail older persons with medical and cognitive co-morbidities has been based largely on extrapolation from studies of younger, healthier research subjects. [35]

Antidepressant medications

Trazodone, buspirone and citalopram were used in studies and patients were found to improve on their depression scores after their use.

Relationship between PSD and other outcomes

Mortality

Depression diagnosed in the acute stage of stroke is predictive of an increased subsequent mortality risk. [37]

Depression and functional recovery after stroke

Depression exerts a detrimental effect on recovery through associated fatigue, lack of hope and lessened motivation for active participation in rehabilitation. [38] Schubert has reviewed this association noting that findings are inconsistent.

Cognitive status

Depression does not cause cognitive impairment but cognitive impairment increases the likelihood of depression[39].

MATERIALS AND METHODS

Hundred subjects aged 60 and above with a first – ever ischemic stroke were randomly selected for the study in the outpatient wing of the Department of Geriatric Medicine, Madras Medical College, Chennai for a period of two years from July 2005 to June 2007.

Informed consent was obtained from all the patients who were interested in the study.

Persons were examined four weeks after the stroke up to a period of two years. The patients had no evidence of premorbid central nervous system disease and did not have cerebral atrophy on CT scan. All were right handed. All patients were able to be interviewed reliably and did not have language problems at the time of interview.

Demographic information like age, sex and information regarding the presence or absence of risk factors such as, hypertension, diabetes mellitus, cardiac disease were obtained in a standardized form. Patients with past personal or family history of depression, parkinsons disease, dementia, previous stroke, other neurological illness, negative stressful life events, living alone, alcohol use were excluded from the study.

A complete physical and neurological examination was carried out. Depression was screened for by using the Geriatric Depression scale 15 -items short version, which assessed symptoms during the past one week. Patients who were found to be depressed were confirmed to have depression by applying DSM IV-TR criteria.

A CT Scan was done in all the patients to localize the infarct site.

Lesions involving the left and right frontal, parietal, temporal, occipital and subcortical regions were compared with the occurrence of PSD. Comparisons were made with the Pearson chi-square statistic.

THE GERIATRIC DEPRESSION SCALE (GDS)

Short Form

Choose the best answer for how you have felt ever the past week :

1. Are you basically satisfied with your life? Yes / **No**.

2. Have you dropped many of your activities and interests? **Yes** / No.

3. Do you feel that your life is empty? **Yes** / No

4. Do you often get bored? **Yes** / No

5. Are you in good spirits most of the time? Yes / **No**

6. Are you afraid that something bad is going to happen to you? **Yes** / No

7. Do you feel happy most of the time? Yes **No**

8. Do you often feel helpless? **Yes** / No

9. Do you prefer to stay at home, rather than going out and doing new things? **Yes** / No

10. Do you feel you have more problems with memory than most? **Yes /** No.

11. Do you think it is wonderful to be alive now? Yes **/ No**

12. Do you feel pretty worthless the way you are now? **Yes** / No

13. Do you feel full of energy? Yes / **No**

14. Do you feel that your situation is hopeless? **Yes** / No.

15. Do you think that most people are better off than you are? **Yes** / No

Answers in bold indicate depression. Score 1 point for each bolded answer.

Scores of 0-4 are considered normal, depending on age, education and complaints.

5.8 Indicate mild depression

9.11 Indicate moderate depression and

12-15 indicate severe depression

The Geriatric Depression Scale (GDS) first created by Yesavage, et al. has been tested and used extensively with the older population. The GDS long form is a brief, 30-item questionnaire in which participants are asked to respond by answering yes or no in reference to how they felt over the past

week. A short form GDS consisting of 15 questions was developed in 1986. Questions from the long form GDS which had the highest correlation with depressive symptoms in validation studies were selected for the short version. Of the 15 items, 10 indicated the presence of depression when answered positively, while the rest (question numbers 1, 5, 7, 11, 13) indicated depression when answered negatively. Scores of 0-4 are considered normal, depending on age, education and complaints; 5-8 indicate mild depression, 9-11 indicate moderate depression and 12-15 indicate severe depression.

The short form is more easily used by physically ill and mildly to moderately cognitively impaired elder adults who have short attention spans and or feel easily fatigued. It takes about 5-7 minutes to complete.

The GDS was found to have a 92% sensitivity and a 89% specificity when evaluated against diagnostic criteria. The validity and reliability of the tool have been supported through both clinical practice and research. In a validation study comparing the long and short forms of the GDS for self rating of symptoms of depression, both were successful in differentiating depressed from non-depressed adults with a high correlation ($r=0.84$, $p<0.001$) (Sheikh and Yesavage, 1986).

The GDS is not a substitute for a diagnostic interview by mental health professionals. It is a useful screening tool in the clinical setting to facilitate assessment of depression in elder adults especially when baseline measurements are compared to subsequent scores. It does not assess for suicidality. The presence of depression warrants prompt intervention and treatment. The GDS may be used to monitor depression over time in all clinical settings. Any positive score above 5 on the GDS short form should prompt an in-depth psychological assessment and evaluation for suicidality.

RESULTS

TABLE - 1

SEX WISE DISTRIBUTION

Sex	DEPRESSION				Total	P value
	No	Mild	Moderate	Severe		
Male	22 (75.9%)	3 (10.3%)	2 (6.9%)	2 (6.9%)	29 (29%)	0.029
Female	31 (43.7%)	13 (18.3%)	18 (25.4%)	9 (12.7%)	71 (71%)	

Among the 100 elderly people, 24% of males and 56% of females were depressed. Table 1 shows this.

Depression was more common in the female population. There was a statistical significance between female population and PSD (P=0.029).

TABLE – 2

AGE WISE DISTRIBUTION

Age in Years	DEPRESSION				Total	P value
	No	Mild	Moderate	Severe		
60-64	20 (50%)	11 (27.5%)	7 (17.5%)	2 (5%)	40 (40%)	0.003
65-70	18 (42.9%)	3 (7.1%)	13 (31%)	8 (19%)	42 (42%)	
> 70	15 (83.3%)	2 (11.1%)		1 (5.6%)	18 (18%)	

Among the 100 elderly people with ischemic stroke and PSD, 20 were in the 60-64 age group, 24 were in the 65-70 age group and 3 were in the > 70 age group. This is shown in table 2. 50% of people in the group 60-64 were depressed, 57% of people in the group 65-70 were depressed and 16% of people in the above 70 age group were depressed.

Majority of the patients were in the age group 65-70 years. There was a high statistical significance between this age group & PSD (P value = 0.003).

TABLE 3

DURATION FROM STROKE AND GDS

Duration from Stroke (months)	DEPRESSION				Total	P value
	No	Mild	Moderate	Severe		
1-6	26 (50%)	11 (21.2%)	7 (13.5%)	8 (15.4%)	52 (52%)	0.179
7-12	17 (53.1%)	5 (15.6%)	9 (28.1%)	1 (3.1%)	32 (32%)	
13-24	10 (62.5%)		4 (25%)	2 (12.5%)	16 (16%)	

The duration after stroke in the patients ranged from 1-24 months. Among the 100 patients, 16% had mild depression, 20% had moderate depression and 11% had severe depression. Most numbers of patients had a duration from stroke ranging from 1-6 months in the study.

There was no statistically significant association between the duration of stroke and PSD.

TABLE 4

HYPERTENSION AND DEPRESSION

Hypertension	DEPRESSION				Total	P value
	No	Mild	Moderate	Severe		
Present	29 (44.6%)	9 (13.8%)	18 (27.7%)	9 (13.8%)	65 (65%)	0.019
Absent	24 (68.6%)	7 (20%)	2 (5.7%)	2 (5.7%)	35 (35%)	

Hypertension as a risk factor was present in 65 patients. Among them, 45% of patients had no depression while 55% of patients had depression. Also among 35 patients without hypertension, 68% of patients were not depressed while 32% were depressed.

There was a statistical significance found between the occurrence of hypertension and PSD (P=0.019).

TABLE 5

DIABETES MELLITUS AND DEPRESSION

Diabetes mellitus	DEPRESSION				Total	P value
	No	Mild	Moderate	Severe		
Present	6 (35.3%)	5 (29.4%)	6 (35.3%)		17 (17%)	0.043
Absent	47 (56.6%)	11 (13.3%)	14 (16.9%)	11 (13.3%)	83 (83%)	

The number of cases with and without diabetes mellitus were 17 and 83 respectively. Among the 17 patients with diabetes, 64% of patients were depressed and among the 83 patients without diabetes, 43% of patients were depressed.

There was a statistical significance found between this risk factor and PSD (P = 0.043).

TABLE 6

CARDIAC DISEASE AND DEPRESSION

Sex	DEPRESSION				Total	P value
	No	Mild	Moderate	Severe		
Male	11 (44%)	3 (12%)	8 (32%)	3 (12%)	25 (25%)	0.351
Female	42 (56%)	13 (17.3%)	12 (16%)	8 (10.7%)	75 (75%)	

Cardiac disease as a risk factor was present in 25 patients among whom 56% of patients were depressed. Of the 75 patients who never had cardiac disease 44% were depressed.

There was no statistical significance between cardiac disease and PSD.

TABLE 7

CEREBRAL HEMISPHERE SIDE ON CT SCAN BRAIN AND

DEPRESSION

Side of lesion	DEPRESSION				Total	P value
	No	Mild	Moderate	Severe		
Left	22 (42.3%)	5 (9.6%)	17 (32.7%)	8 (15.4%)	52 (52%)	0.001
Right	31 (64.6%)	11 (22.9%)	3 (6.3%)	3 (6.3%)	48 (48%)	

The cerebral hemisphere wise distribution shows that among 52 persons with left sided lesions on the CT scan, 57% of them were depressed. Of the 48 persons with right sided lesions, 35% of them were depressed.

52% of the patients in the study had left sided infarcts on CT scan brain and 48% of the patients had right sided infracts on CT scan brain. There was a high statistical significance between left sided lesions and PSD (P-0.001).

TABLE 8

SITE OF LESION ON CT SCAN BRAIN ON LEFT SIDE AND DEPRESSION

Site	DEPRESSION				Total	P value
	No	Mild	Moderate	Severe		
Normal Study	2 (50%)	2 (50%)			4 (7.7%)	0.00839
Frontal			5 (62.5%)	3 (37.5%)	8 (15.4%)	
Temporal	3 (60%)		2 (40%)		5 (9.6%)	
Parietal	7 (100%)				7 (13.5%)	
Occipital	1 (100%)				1 (1.9%)	
Ganglio capsular	9 (33.3%)	3 (11.1%)	10 (37%)	5 (18.5%)	27 (51.9%)	

Among left sided lesions the distribution shows that 8 persons with PSD had frontal lobe infarct, 100% of whom were depressed. 2 had temporal infarct, 40% of whom were depressed and 18 had gangliocapsular infarcts,66% of whom were depressed.

There was a highly significant statistical association between lesions in frontal lobe and gangliocapsular region infarcts and PSD (P=0.008).

TABLE 9

SITE OF LESION ON CT SCAN BRAIN ON RIGHT SIDE AND DEPRESSION

Site	DEPRESSION				Total	P value
	No	Mild	Moderate	Severe		
Normal Study	1 (100%)				1 (2.1%)	0.006
Frontal	2 (33.3%)		2 (33.3%)	2 (33.3%)	6 (12.5%)	
Parietal	6 (40%)	7 (46.7%)	1 (6.7%)	1 (6.7%)	15 (31.3%)	
Temporal	3 (100%)				3 (6.3%)	
Occipital		1 (10%)			1 (2.1%)	
Gangliocapsular	19 (86.4%)	3 (13.6%)			22 (45.8%)	

Among right sided lesion, 66% of persons with frontal infarcts were depressed, 60% of persons with parietal infarcts were depressed, 1 patient with occipital infarct was depressed and 13% of patients with gangliocapsular infarcts were depressed.

There was a highly significant statistical association between lesions in frontal and parietal regions and PSD (P-0.006).

DISCUSSION

Depression is the most common emotional disorder following stroke. The risk seems to be greatest within the first 2 years following a cerebrovascular accident, when major depression develops in about 20% and minor depression in another 20%. If left untreated, major depression seems to last for approximately 8-9 months according to some studies. This study seeks to find out if there exists a correlation between PSD and inter hemispheric and intra hemispheric lesion location.

This study is a cross sectional observational study. So it has its own limitations. Cross sectional studies are susceptible to several sources of bias and confounding.

Dynamic changes are known to occur in the relations between poststroke mood disorders and associated variables during follow up. Left hemisphere lesion location had the strongest correlation with the severity of depression for the first year following stroke. Performance at a single point in time may bear little relation to subsequent changes in performance over time. Longitudinal studies that measure the severity of depression over time greatly reduces bias and are preferred but they are much more expensive and subject to their own sources of bias.

In this study, only patients without aphasia or comprehension deficits were included. Only patients who were physically able to walk to the outpatient clinic were selected. Not all patients with clearly evident clinical stroke had positive brain CT scans at the time of the study .

PSD was assessed by applying the Geriatric Depression Scale – short version and subsequently confirmed by the DSM IV-TR criteria. Those patients who had previous history of depression, with

family history of depression, with spouse dead and living alone, with past history of suicide attempts were not included in the study as they would independently alter the occurrence of poststroke depression. But other medical conditions like hypertension, diabetes, cardiac disease and smoking could not be excluded as they were the risk factors for stroke in the elderly. Though each of these conditions could themselves influence the occurrence of PSD, they could not be eliminated.

The mean age of the study group was from 60-85 years. The duration of stroke was from one month to twenty four months. The occurrence of depression as assessed by the Geriatric Depression scale was found to be statistically significant with the female sex. This is consistent with previous studies[29,40].

Age was found to have significant relationship with PSD[41]. The study found no significant association between the duration of stroke and PSD.

There was a statistically significant association found to exist between hypertension, diabetes and PSD. No relationship was found between cardiac disease and PSD. In the study, infarcts situated in the left cerebral hemisphere had a significant association with PSD. Moreover frontal and gangliocapsular region infarcts were highly significantly associated with PSD.[11,20,24].

Among those with right hemisphere stroke lesions, these with frontal and parietal damage showed the highest frequency of depression[22,23]. Longitudinal studies have found that proximity of the lesion to frontal pole is significantly associated with the severity of depression most strongly during the first six months poststroke, suggesting that this phenomenon is a dynamic one that changes over time.

CONCLUSION

1. There is a significant association between PSD and left hemispheric infarcts.

2. There is a significant association between left frontal lobe infarcts and left gangliocapsular region infarcts and PSD. There is a significant association between right frontal and parietal damage and PSD.

3. Risk factors hypertension and diabetes were found to be significantly associated with PSD.

4. There was no significant association between the severity of PSD and the duration from stroke.

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PROFORMA

Name : Serial No.

Age :

Sex :

Out Patient No. :

Socio-economic status

Clinical Diagnosis

Duration of stroke

History of Risk Factors

Hypertension

Diabetes Mellitus

Cardiac Disease

Examination

PR

BP

Cardiovascular system

Respiratory system

Abdomen

Neurological examination

- Side of stroke
- Extent of weakness
- Speech

Geriatric Depression Scale

Investigations

Urine

Blood TC

DC

ESR

Hb

	Sugar
	Urea
	Creatinine
Chest X-Ray	
ECG	
Echo cardiogram	
CT Scan	Side
	Lesion
	Site

MASTER CHART

S.No.	Age	Sex	HT	DM	Cardiac disease	GDS	Side of Lesion	Site of Lesion	Duration from stroke
1	75	1	2	2	1	2	1	Temporal	12 months
2	67	2	2	1	2	3	1	Normal study	4 months
3	76	2	1	2	2	6	1	Normal study	3 months
4	79	2	2	2	2	2	1	Gangliocapsular	24 months
5	65	2	1	1	1	11	1	Gangliocapsular	24 months
6	70	2	2	2	1	2	1	Gangliocapsular	8 months
7	85	2	2	2	2	3	1	Temporal	12 months
8	69	2	1	2	1	9	1	Gangliocapsular	12 months
9	63	2	1	2	1	1	1	Temporal	9 months
10	63	1	1	2	2	3	1	Gangliocapsular	8 months
11	65	2	1	2	2	10	1	Gangliocapsular	20 months
12	65	2	1	2	1	12	1	Frontal	20 months
13	62	2	1	2	2	6	1	Gangliocapsular	3 months
14	64	2	1	2	2	13	1	Frontal	4 months
15	64	2	1	2	1	10	1	Parietal	12 months
16	65	2	1	2	2	11	1	Parietal	12 months
17	63	2	1	1	1	9	1	Frontal	3 months
18	70	2	1	2	1	10	1	Gangliocapsular	7 months
19	60	2	1	2	2	2	1	Gangliocapsular	4 months
20	63	2	1	2	2	9	1	Gangliocapsular	9 months
21	65	1	1	1	1	2	1	Temporal	15 months
22	74	2	1	2	1	1	1	Gangliocapsular	3 months
23	70	2	2	2	2	3	1	Gangliocapsular	8 months
24	69	2	1	2	2	14	1	Gangliocapsular	4 months
25	60	2	1	2	2	9	1	Gangliocapsular	4 months
26	67	1	2	2	1	12	1	Gangliocapsular	4 months
27	76	1	1	2	1	1	2	Gangliocapsular	14 months
28	60	2	2	2	2	2	2	Gangliocapsular	3 months
29	65	1	2	1	2	11	2	Frontal	2 months
30	72	1	1	2	2	4	2	Gangliocapsular	4 months
31	61	2	1	2	1	5	2	Occipital lobe	12 months
32	60	2	2	2	1	6	2	Parietal	3 months
33	65	2	1	2	2	3	2	Parietal	3 months
34	70	1	2	1	2	6	2	Parietal	5 months
35	65	2	1	2	2	13	2	Frontal	4 months

S.No.	Age	Sex	HT	DM	Cardiac disease	GDS	Side of Lesion	Site of Lesion	Duration from stroke
36	65	2	1	2	1	2	2	Temporal	9 months
37	60	2	1	2	2	5	2	Gangliocapsular	12 months
38	60	2	2	2	2	3	2	Parietal	4 months
39	70	2	2	2	2	2	2	Gangliocapsular	24 months
40	63	1	1	2	2	1	2	Gangliocapsular	8 months
41	60	2	2	1	2	8	2	Parietal	3 months
42	60	1	1	2	2	2	2	Gangliocapsular	5 months
43	72	1	1	2	2	1	2	Temporal	3 months
44	64	1	1	2	2	3	2	Gangliocapsular	5 months
45	60	1	2	1	2	4	2	Gangliocapsular	6 months
46	65	1	2	2	2	3	2	Parietal region	6 months
47	60	2	2	2	2	2	2	Gangliocapsular	24 months
48	77	2	1	2	1	4	2	Frontal	12 months
49	70	2	1	2	2	10	2	Frontal	1 month
50	75	1	2	2	1	2	1	Temporal	12 months
51	67	2	2	1	2	3	1	Normal study	4 months
52	76	2	1	2	2	5	1	Normal study	3 months
53	78	2	2	2	2	2	1	Occipital	24 months
54	65	2	1	1	1	9	1	Gangliocapsular	24 months
55	70	2	2	2	2	1	1	Parietal	8 months
56	79	2	2	2	2	2	1	Parietal	12 months
57	69	2	1	2	1	10	1	Gangliocapsular	12 months
58	63	2	1	2	2	1	1	Temporal	9 months
59	63	1	1	2	2	2	1	Gangliocapsular	8 months
60	65	2	1	2	2	11	1	Gangliocapsular	20 months
61	65	2	1	2	2	13	1	Gangliocapsular	20 months
62	62	2	1	2	2	5	1	Gangliocapsular	3 months
63	64	2	1	2	2	15	1	Gangliocapsular	4 months
64	64	2	1	2	1	11	1	Frontal	12 months
65	65	2	1	2	2	10	1	Frontal	12 months
66	63	2	1	1	2	9	1	Frontal	3 months
67	70	2	1	2	2	11	1	Gangliocapsular	7 months
68	60	2	1	2	2	3	1	Gangliocapsular	4 months
69	63	2	1	2	2	8	1	Gangliocapsular	9 months
70	65	1	1	1	2	1	1	Temporal	15 months
71	74	2	1	2	1	2	1	Gangliocapsular	3 months
72	70	2	2	2	2	1	1	Parietal	8 months
73	69	2	1	2	2	14	1	Gangliocapsular	4 months

S.No.	Age	Sex	HT	DM	Cardiac disease	GDS	Side of Lesion	Site of Lesion	Duration from stroke
74	60	2	1	2	2	11	1	Frontal	4 months
75	67	1	2	2	2	13	1	Frontal	4 months
76	76	1	1	2	2	1	2	Gangliocapsular	14 months
77	60	2	2	2	2	2	2	Gangliocapsular	3 months
78	65	1	2	1	2	10	2	Parietal	2 months
79	72	1	1	2	2	3	2	Gangliocapsular	4 months
80	61	2	1	2	2	7	2	Parietal	12 months
81	60	2	2	2	1	6	2	Parietal	3 months
82	65	2	1	2	2	1	2	Parietal	3 months
83	70	1	2	1	2	5	2	Parietal	5 months
84	65	2	1	2	2	4	2	Normal study	4 months
85	65	2	1	2	1	3	2	Temporal	9 months
86	60	2	1	2	2	7	2	Gangliocapsular	12 months
87	60	2	2	2	2	2	2	Gangliocapsular	4 months
88	70	2	2	2	2	1	2	Gangliocapsular	24 months
89	63	1	1	2	2	3	2	Gangliocapsular	8 months
90	60	2	2	1	2	5	2	Gangliocapsular	3 months
91	60	1	1	2	2	1	2	Gangliocapsular	5 months
92	72	1	1	2	2	2	2	Parietal	3 months
93	64	1	1	2	2	1	2	Gangliocapsular	5 months
94	60	1	2	1	2	3	2	Gangliocapsular	6 months
95	65	1	2	2	2	1	2	Parietal region	6 months
96	60	2	2	2	2	1	2	Gangliocapsular	24 months
97	77	2	1	2	1	13	2	Parietal	12 months
98	70	2	1	2	2	3	2	Frontal	1 month
99	70	1	2	1	2	5	2	Parietal	5 months
100	65	2	1	2	2	13	2	Frontal	4 months

Sex
Female-1
Male - 2

Risk Factors
Present - 1
Absent - 2

Age
60 -64 years - 1
65-69 years -2
70-74 years -3
75-79 years -4

Side of Lesion
Left - 1
Right - 2

Duration
0-6 months - 1
6-12 months - 2
12-18 months - 3
18-24 months - 4

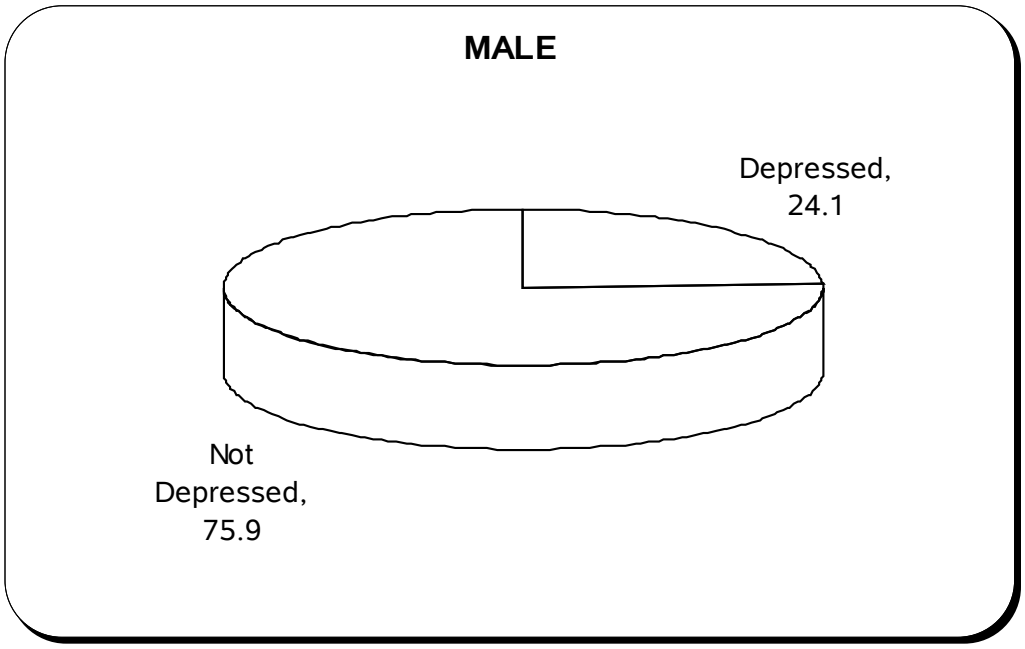
Site of Lesion
Normal study -1
Frontal -2
Gangliocapsular -3
Temporal - 4
Parietal -5

GDS
1-4 - No Depression -1
5-8 - Mild Depression - 2
9-11 - Moderate Depression - 3
12-15 - Severe Depression -4

GRAPHS

Fig. 1

SEX WISE DISTRIBUTION



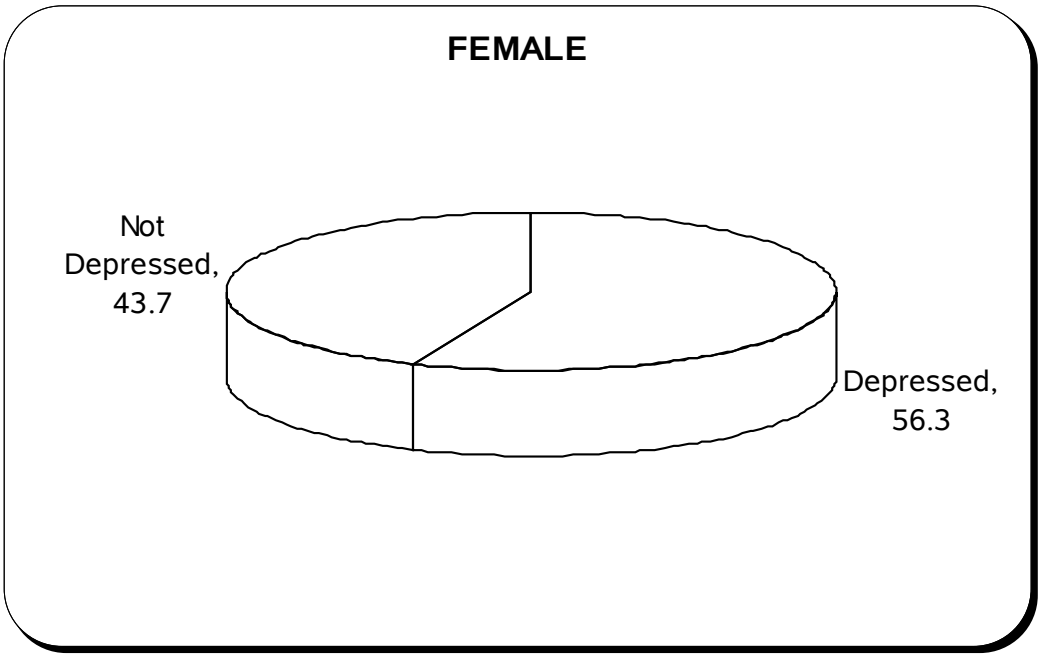

FEMALE
Not Depressed, 43.7
Depressed, 56.3

Fig. 2

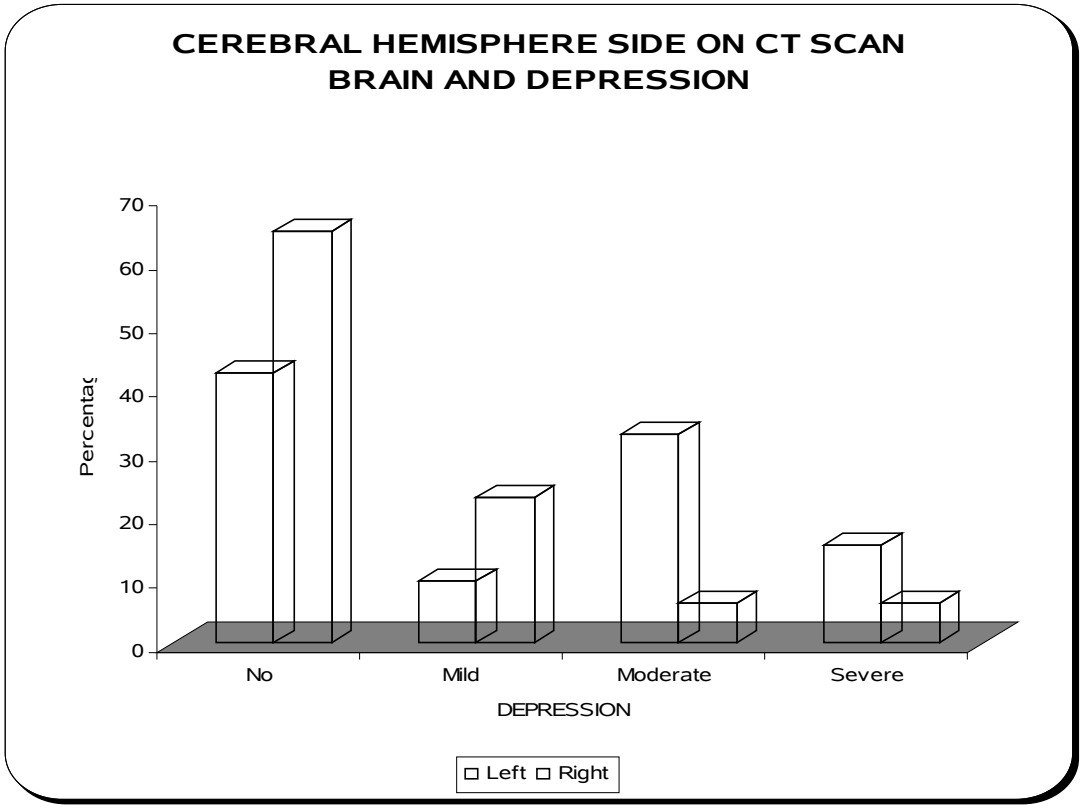

CEREBRAL HEMISPHERE SIDE ON CT SCAN
BRAIN AND DEPRESSION
70
60
50
40
30
20
10
0
No
Mild
Moderate
Severe
DEPRESSION
Left
Right

Fig. 3

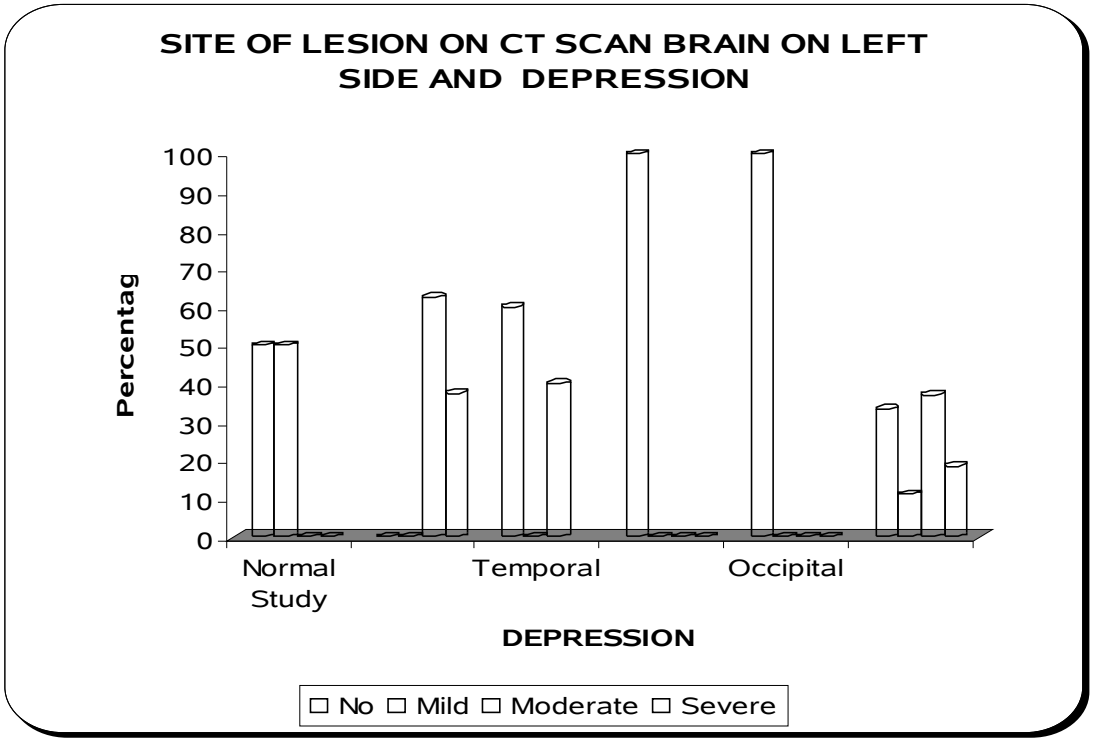
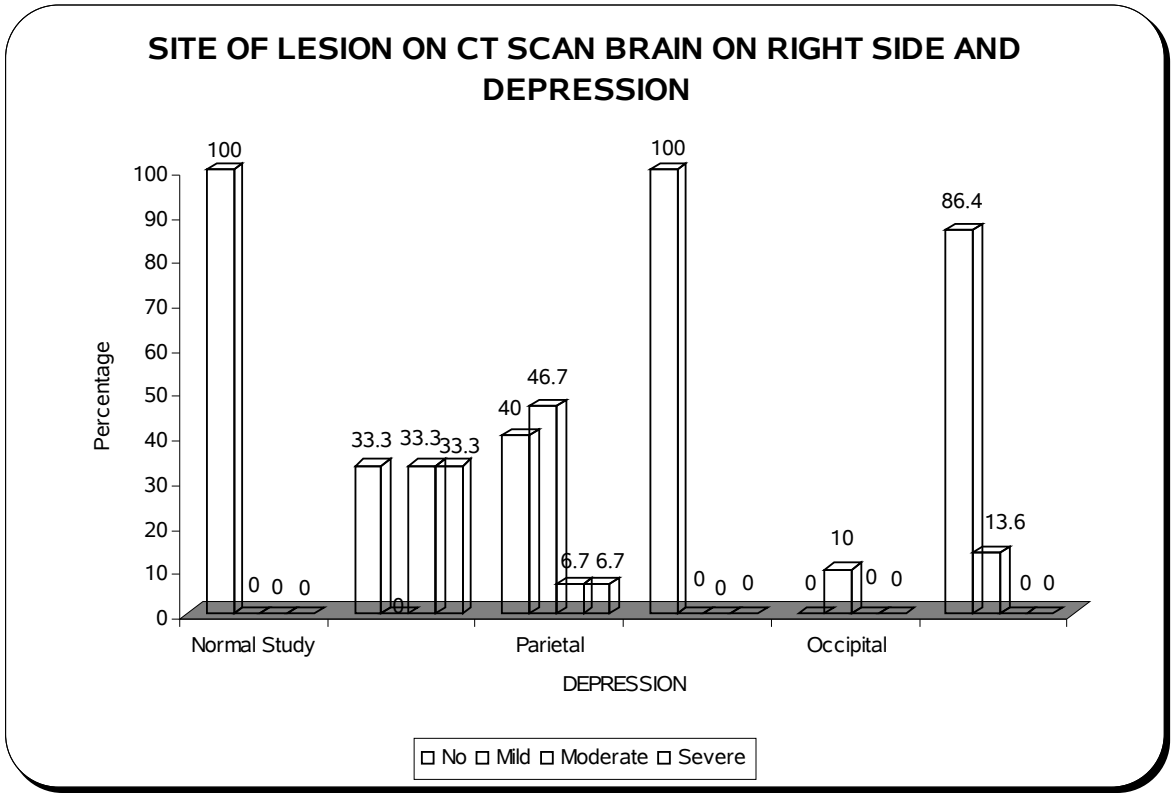

SITE OF LESION ON CT SCAN BRAIN ON LEFT SIDE AND DEPRESSION
Percentag
100
90
80
70
60
50
40
30
20
10
0
Normal Study
Temporal
Occipital
DEPRESSION
No
Mild
Moderate
Severe

Fig. 4


SITE OF LESION ON CT SCAN BRAIN ON RIGHT SIDE AND DEPRESSION
Percentage
100
90
80
70
60
50
40
30
20
10
0
100
0 0 0
33.3 33.3 33.3
0
40
46.7
6.7 6.7
100
0 0 0
0
10
0 0
86.4
13.6
0 0
Normal Study
Parietal
Occipital
DEPRESSION
No Mild Moderate Severe